AMENDMENTS TO THE SPECIFICATION:

Please amend the specification as follows:

Please replace the paragraph beginning at line 7 on page 4 with the following amended paragraph:

--(a) the nucleotide sequence as set forth in residues 73 to ~~601~~ 582 in SEQ ID NO:1;--

Please replace the paragraph beginning at line 11 on page 4 with the following amended paragraph:

--(c) the nucleotide sequence as set forth in residues 73 to ~~451~~ 432 in SEQ ID NO:1;--

Please replace the paragraph beginning at line 15 on page 4 with the following amended paragraph:

--(e) the nucleotide sequence as set forth in residues ~~485~~ 466 to ~~820~~ 801 in SEQ ID NO:1;--

Please replace the paragraph beginning at line 18 on page 98 with the following amended paragraph:

--**Apo-A-I is the HS inhibitor of T cell-signaling of monocytes.** To determine whether apo-A-I displayed the inhibitory activity, commercially available, purified apo-A-I (Sigma Fine Chemicals, St. Louis, MO) was tested. Apo-A-I inhibited the production of both TNF-α and IL-1β in THP-1 cells activated by membranes of HUT-78 cells in a dose-dependent manner (Fig. ~~6A~~ 7A). As already evident from Fig. 3, TNF-α production was less inhibited than IL-1β production. Since the apo-A-I preparation contained 3% unidentified contaminants (according to the supplier), it had to be ascertained that the

inhibition was indeed due to apo-A-I. Proteins from delipidated HDL were subjected to preparative SDS-PAGE. After copper-~~straining~~staining, bands (M_r: 56,000-66,000, 50,000, 28,000, and 18,000) were excised and electroeluted. The inhibitory activity was recovered in the M_r =28,000 and M_r =18,000 bands (Fig. ~~6~~ 7, B and C). The double M_r =56,000-66,000 band, which contained apo-A-I aggregates as assessed by Western blotting, did not show significant inhibitory activity (not shown). Production of both IL-1β and TNF-α was inhibited by the electroeluted proteins. TNF-α was inhibited to a lower extent than IL-1β, confirming the results from Fig. 2. All inhibitory fractions contained apo-A-I as demonstrated by Western blot analysis (Fig. ~~6E~~ 7E), pointing to apo-A-I as the inhibitor of T cell-signaling of monocytes. Indeed, it is very unlikely that another HDL apolipoprotein would display the same behavior as apo-A-I in terms of size of protein and proteolytic fragment (Fig. ~~6E~~ 7E, lanes a and b). Alternatively, proteins from delipidated HDL were subjected to gel filtration on Superdex S75. Pooled fractions corresponding to M_r = 28,000 ± 10,000 displayed the inhibitory activity (Fig. ~~6D~~ 7D). These fractions contained apo-A-I as determined by Western blot analysis (Fig. ~~6E~~ 7E, lane c), further confirming that apo-A-I was the inhibitor.--

Please replace the paragraph beginning at line 10 on page 99 with the following amended paragraph:

--**HDL interact with stimulated T cells through apo-A-I binding.** To determine whether the inhibitory activity of HDL was due to its potential binding to stimulated T cell membranes or to THP-1 cells, either THP-1 cells or membranes isolated from stimulated HUT-78 cells were preincubated in the presence or absence of FCS, HS, or isolated HDL. After washing, the residual activation capacity of membranes from

stimulated HUT-78 cells on THP-1 cells was assessed. The inhibition of IL-1β production was observed only when membranes of stimulated HUT-78 cells were incubated with HS or HDL (Fig. ~~7A~~ 6A). Incubation of THP-1 cells with either FCS, HS, or HDL did not appear to inhibit the production of IL-1β (Fig. ~~7A~~ 6A). These results demonstrate that the inhibitory activity of HS and HDL was mainly directed to the activating factor(s) expressed at the surface of stimulated T cells.--

Please replace the paragraph beginning at line 21 on page 99 with the following amended paragraph:

--To further confirm that the inhibitory factor(s) interacted with surface factors on stimulated T cells, isolated HDL was labeled with fluorescein isothiocyanate (FITC) and its binding to different cell types assessed by flow cytometry. No binding of FITC-HDL was observed on THP-1 cells (Fig. ~~7B~~ 6B), whereas fluorescence of monocytes was slightly enhanced when incubated with FITC-HDL as compared to unconjugated FITC control (Fig. ~~7C~~ 6C). A low level of binding of FITC-HDL to unstimulted HUT-78 cells was observed, whereas stimulated HUT-78 cells bound FITC-HDL, displaying 2 fluorescent peaks, suggesting the presence of at least 2 different HDL binding sites (Fig. ~~7~~ 6, D and E). At a lower FITC-HDL concentration, only one fluorescent peak was observed. In the presence of anti-apo-A-I antibodies, a shift toward lower fluorescence intensity was observed, demonstrating that HDL interacted with stimulated T cells via apo-A-I specific binding (Fig. ~~7F~~ 6F). Together these results show that HDL interacted preferentially with stimulated T cells, implying that the inhibitory activity involving apo-A-I was directed to surface factors on T cells.--